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An Overlooked Complication of Vancomycin Induced Acute Flaccid Paralysis in a Child with Acute Leukemia: A Case Report

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Abstract

Vancomycin is a glycopeptide antibiotic which is commonly used to treat methicillin-resistant staphylococcal infections. It is commonly used in pediatric oncology wards for children with febrile neutropenia. We report a very rare side effect of vancomycin induced myopathy in a child with acute lymphoblastic leukemia. To the best of our knowledge, this is the first case reported from Oman.

Keywords: Vancomycin; Flaccid Paralysis; Acute Leukaemia.

Introduction

Children with leukemia who develop lower motor neuron lesion manifested as hypotonia, hyporeflexia or diminished power are usually diagnosed to have peripheral neuropathy due to vincristine; a vinca alkaloid chemotherapeutic agent used in all phases of therapy.[1] Vancomycin is a glycopeptide antibiotic commonly used to treat methicillin-resistant staphylococcal infections.[2] In the field of oncology, it is widely used in the treatment of infections in patients with febrile neutropenia. The common adverse effects of vancomycin include red man syndrome, renal toxicity, ototoxicity, cardiovascular depression and anaphylactoid reaction.[3] There are reported cases of potentiation of neuromuscular blockage, mononeuritis multiplex, neuralgic amyotrophy and peripheral neuropathy after vancomycin infusion.[4,5,6,7] We report a case of a child with acute lymphoblastic leukemia, who developed vancomycin-induced lower motor neuron lesion, with intact nerve conduction studies.

Case Report

A five year old boy was diagnosed to have pre-B ALL in September 2010 and was started on UK ALL 2003 high risk protocol (Reference). On the 11th week of chemotherapy, the child developed febrile neutropenia (temperature 39^{o}C; absolute neutrophil count of 0.1x10^{9}/l) and was started on tazobactam-pipracilline and gentamycin. After two days, he developed perianal cellulitis. Therefore, vancomycin was added for coverage of coagulase-negative staphylococci and staphylococcus aureus. Blood cultures remained negative. After starting vancomycin infusion, the child developed itching, erythematous rash on the face, neck and limbs (Redman syndrome), thus the infusion time was increased. However, the symptoms persisted and the infusion was consequently stopped. Two hours later, the child was reported by the mother to have generalized body aches, muscle pains and lower limbs weakness. On examination, he was found to have severe hypotonia, areflexia of knees and ankles with a flexor planter response. All muscle groups of lower limbs had a power of 2/5, indicating lower motor neuron lesion. There was also tenderness of all muscle groups, suggesting a muscle disease, however, sensations were intact and sphincters control was preserved.

Investigations on electrolytes, bone profile, blood culture, urine culture, and creatine phosphokinase, as well as nerve conduction study were performed. Unfortunately, the electromyogram was not performed. The child continued to be symptomatic with gradual recovery, and regained his normal gait on the fifth day of discontinuation of vancomycin.

Discussion

We report a case of vancomycin-induced acute flaccid paralysis in a child with ALL. In this case, the flaccid paralysis was attributed to vancomycin as the child developed the symptoms after receiving first dose of vancomycin and the symptoms ameliorated once the drug was stopped. Vancomycin induced peripheral neuropathy, mononeuritis multiplex and neurologic myotrophy were excluded in our patient as the nerve conduction study was normal. Of interest, the child developed Redman syndrome, which was aggravated on re-exposure of the drug. It is well known that this side effect is a hypersensitivity reaction to the drug associated with histamine release.[8] It is also well established that histamine release may be associated with other manifestations such as hypotension, however the association with acute flaccid paralysis has never been reported.

Another common cause of acute flaccid paralysis in children with ALL, is the vincristine induced peripheral neuropathy.[9,10] Again, this was excluded for the same reason and the fact that the child received vincristine more than three weeks before the onset of symptoms.

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There are few reported cases in the literature of gentamicin induced muscle weakness,[11] however, it is unlikely in our case as the child was exposed to gentamicin previously (4 times) with no similar problems. Moreover, there are reports that combination of vancomycin and gentamicin can induce neuromuscular blockage (NMB).[12,13] It is possible in our case that they worked together and resulted in the severe clinical picture. However, there is no confirmatory test to prove neuromuscular junction blockage and the clinical picture was not typical of NMB.[14]

In the current case, there was generalized muscle pains and tenderness that usually suggest a muscle disease and myositis; however the creatinine kinase levels were normal, excluding the possibility of myositis but not the myopathy. Unfortunately, the EMG which is more sensitive for muscle disease was not performed for technical reasons. In the current reported case, the most likely explanation for the patient's presentation is vancomycin induced myopathy severe enough to cause a lower motor neuron manifestations mounting to severe paralysis.

Conclusion

In summary, we report the first case of vancomycin induced myopathy in Oman. Although it is a very rare side effect, the clinicians treating children with vancomycin should be aware of it.

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